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THE
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THE SIGNIFICANCE OF A POSITIONAL NYSTAGMUS IN OTONEUROLOGICAL DIAGNOSIS.*

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Chicago, Ill.

The term "positional nystagmus" refers to a nystagmus which appears only during certain positions of the head or which is greatly influenced by the position of the head. Such a nystagmus accompanies the subjective complaint of vertigo occurring in these certain positions, commonly referred to as postural vertigo. This symptom with its accompanying nystagmus is known by both animal experimentation and clinical observation to indicate a disturbance in the region of the vestibular sense organs or the central pathways therefrom and is a frequent occurrence in a variety of affections of the central nervous system as well as an occasional accompaniment of a peripheral ear disturbance.

The present report is chiefly concerned with, first, the evaluation of positional nystagmus as a means of differentiation between a peripheral and central origin of the disturbance and its possible application in clinical cases of vertigo which cannot otherwise be localized (sometimes described as pseudo-Ménière's syndrome), and second, on the basis of this means of differentiation, in directing attention to the frequency with which vertigo of this type must be ascribed to a central disturbance, of circulatory or vasomotor nature.

On the basis of the theories of Magnus and de Kleijn and of Quix that the otolith system was concerned with static equi-

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librium, vertigo and nystagmus occurring in certain postures of the head were at first considered to be due to a disturbance of the otoliths and therefore a peripheral ear disease. Experiments with acute alcohol poisoning in dogs by Rothfeld,[1] de Kleyn and Versteegh[2] seemed to substantiate this view.

Later observations by many observers have established the possibility of origin in either the central or peripheral vestibular apparatus.

Experimentally positional nystagmus has been observed by Nylen[3] following centrifuging experiments and introduction of foreign material into the vestibule, also by Chilow[4] as a temporary stage of a labyrinthine reaction following introduction of irritative substances into the niches of the round and oval windows and by radium emanation. Fromm and Nylen[5] have observed positional nystagmus after implantation of sarcoma in the posterior cranial fossa, both in the labyrinthectomized animals and in those with intact labyrinths. Acute quinine poisoning in dogs has been found by Seiferth[6] to produce a positional nystagmus, probably of central origin.

Clinically, the diagnostic value of positional nystagmus in brain tumors has been evaluated carefully by Nylen,[7] who found it to be present in about 90 per cent of posterior fossa tumors, in comparison with a spontaneous nystagmus in less than 60 per cent.

Nylen classified positional nystagmus into two types:

Type I. The direction of the nystagmus changes with a change in position of the head.

Type II. The direction of the nystagmus does not change with changes in position, but may appear only in certain positions or may be influenced in intensity by the position of the head.

Type I was found to occur predominantly in cerebellar and pontine tumors, while type II was usually found in cerebellopontine angle tumors.

The impression was gained by him, also, that a vertical

nystagmus downward occurring in the hanging head position was strong evidence of a supratentorial location of the tumor.

A series of observations have been reported by Seifert[6] on the occurrence of positional nystagmus in otological and neurological conditions other than tumors. The classification used was similar except that under type I was specified both positional nystagmus which was "irregular" as to occurrence, direction and intensity, and one which regularly altered in direction with change of position. His observations indicated that, having excluded a labyrinthine fistula, a type I positional nystagmus was typical for centrally located disturbances. He attempted to make a further differentiation between intracerebral lesions such as tumors and other intracranial lesions such as concussion on the basis of the positional nystagmus (type I) being of the "irregular" or "regular" variety. The occurrence of these two varieties was related by him to the duration of the pressure fluctuations or circulatory disturbances affecting the vestibular pathways.

Type II, or positional nystagmus of constant direction, was found by him in a limited number of peripheral ear disturbances as well as in some centrally located lesions such as trauma and concussion.

Ruttin,[8] whose classification varied somewhat from the above, also expressed the opinion that a positional nystagmus without regularity of direction indicated a centrally located lesion.

The clinical material which forms the basis of this report has been observed during a period of approximately 12 years. During that time the examination for positional nystagmus was not made in all cases of vertigo, but a series of approximately 100 cases, exclusive of labyrinthitis and known brain lesions, has been personally checked for positional nystagmus as part of the routine examination. Of this group, approximately two-thirds were diagnosed as Ménière's disease on the basis of recurrent attacks of vertigo associated with tinnitus and deafness. Positional nystagmus was demonstrated infrequently in the Ménière's disease group, only four cases

being observed, although on the basis of the history it was evident that a postural vertigo had existed as a temporary phase following the acute stage of the attack, in a greater number. The fact that the great majority of cases of Ménière's disease have been seen after the vertigo attack has passed off probably prevented a more frequent observation of positional nystagmus.

One-third of the group consisted of cases of vertigo which were not associated with auditory symptoms and in which no associated signs of disease of the central nervous system could be demonstrated, sometimes called pseudo-Ménière's syndrome. It was this group in which the value of the positional nystagmus as a localizing sign was of particular interest and which forms the chief topic of discussion in this report. In this group the vertigo was sometimes severe and present in all positions during the initial stages of the attack, possibly up to two or three days, then disappearing except when certain postures were assumed. In some instances, usually in attacks of lesser severity, the vertigo was of a postural character from the onset. The duration varied from days to several weeks.

In contrast to Ménière's disease and other known peripheral lesions, the postural character of the vertigo was almost a constant finding in this group, although sometimes obscured during the height of the attack. The tests for vestibular function were routinely normal. The attacks occurred at all age periods from the third to the seventh decade and the duration was from a few days to several weeks, sometimes as long as two to three months. Some cases have had recurrences on several occasions within a period of years, but more often the attacks did not recur, or if so, only after long intervals.

METHOD OF EXAMINATION.

The history almost invariably provides an indication as to whether the vertigo is postural in character when a specific inquiry is made. If examined during the period when the complaint is present, the phenomenon could almost invariably be reproduced, thereby confirming the patient's statements and obtaining objective information of diagnostic value. So

clear-cut is the history of postural vertigo that a disturbance of the vestibular system can usually be diagnosed on that basis alone.

It has been routine practice to test for positional nystagmus in all cases in which the history suggested its presence.

The routine tests for labyrinthine and vestibular function in such cases have been as follows:

1. Local ear, nose, throat and larynx examination. Tests for ataxia such as finger-nose test, test for dysdiadokokinesis, Romberg's test and gait.

2. Auditory function. Whispered voice, tuning forks, audiometer and monochord.

3. Spontaneous nystagmus in upright position, or lying position when necessary, both without and with Bartel's or Frenzel's spectacles for elimination of fixation.

4. Head shaking test.

5. Compression over the carotid sinuses.

6. Tests for positional nystagmus.

7. Tests for vestibular excitability. The cold caloric test was routinely done using the spectacles for elimination of fixation, and the head in the 60° backward position. The stimulus used consisted either of 5 cc. of water at 80° F. directed against the drum through a small needle, or to flood the canal with water at 80° F. for 10 seconds. The latter method is simpler to carry out since the head can be tipped slightly to one side during the 10 seconds to keep the water in the canal. A more constant stimulus is thereby delivered.

When the minimal type of stimulus is used, it is essential to eliminate fixation by the use of the glasses to obtain accurate determinations. With a normal response no further test is made. With an abnormal result other tests were sometimes necessary, such as increasing the stimulus, and the warm caloric tests. The rotation and galvanic tests have seldom been found to be of assistance in this group of cases.

ROUTINE METHOD OF EXAMINATION FOR POSITIONAL NYSTAGMUS.

The patient sits upright on the examining table and is observed for nystagmus.

During the following changes of position the attempt is made to keep the head and body in the same relation to avoid neck torsion, thereby avoiding as much as is practically possible any neck reflex or effect of torsion on the cerebral circulation. While it is not possible to avoid some degree of neck torsion without a specially constructed table, the following simple method of testing is satisfactory for clinical purposes.

During the positional tests the eyes are routinely observed for nystagmus in the front gaze position.

The patient is then lowered to the supine position. The movement is made fairly rapidly, since a slow movement will sometimes fail to elicit the symptom. Since an optokinetic nystagmus occurs normally during movement with the eyes open, any labyrinthine response is thereby obscured during movement. A positional nystagmus is, however, only that which is present or appears after the new position has been reached. The eyes are observed for five to 10 seconds in each new position. Positional nystagmus may appear immediately in the new position or may come on slowly, requiring a few seconds to reach a maximum. It may be maintained or, having reached a peak, may then recede in about the same rate and disappear. Occasionally it may reverse in direction without further change of position. It may be irregular in its occurrence, so that a negative response in the presence of positive complaints should not be accepted without repetition of the tests. In this regard the rate or abruptness with which the position is altered may be a factor.

The two lateral positions are then tested, the patient being brought back to the supine position and observed after each new position, since a nystagmus may appear in that position for the first time after it has been brought on in a lateral position. In other words, the appearance of a nystagmus in the supine position may be influenced both by the previous position and the rate at which the alteration takes place.

Following the lateral positions, the head is then extended over the end of the table in the so-called head-hanging position. The head was sometimes turned to right and left in the head-hanging position as well.

The prone position was rarely used because the same head position was more easily obtained by stooping forwards.

The patient was then observed on sitting up from the supine position, on stooping forward and again on raising to the upright position.

The routine tests for positional nystagmus seldom occupied more than five minutes.

The method found to be most convenient for recording the findings included the use of the diagram suggested by Fren-

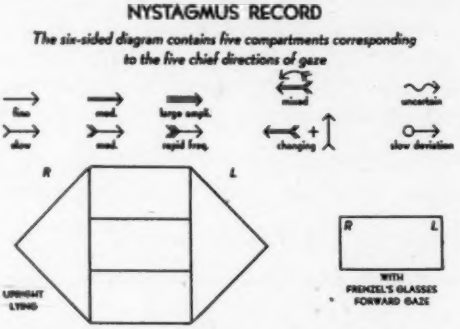

Fig. 1.

zel[9] for spontaneous nystagmus containing five compartments to represent five directions of gaze (see Fig. 1), using an arrow to indicate the type and direction of nystagmus. The number of bars in the shaft of the arrow indicate amplitude and the tail feathers the frequency.

The findings on positional tests were similarly recorded as to character, direction and duration in small rectangles. Additional rectangles may be added when necessary; for example, to indicate a nystagmus in the supine position, following a lateral position.

DESCRIPTION OF CASES.

While the incidence and type of positional nystagmus in brain tumors has been thoroughly investigated by Nylen,[7] and in a number of other otoneurological conditions by Seiferth,[6] it is desirable for illustrative purposes here to describe the findings in representative cases of known etiology, both peripheral and central, as a background for the interpretations which seem to be warranted in those cases where the localization otherwise remained in doubt.

POSITIONAL NYSTAGMUS TYPE II IN PERIPHERAL LESIONS.

Case 1: J. B., male, age 35. Severe vertigo, nausea and vomiting. Unilateral tinnitus and mild low tone deafness came on just before the vertigo. Symptoms subsided gradually and after four days vertigo was present only in certain positions. History of slight dizziness on arising on several occasions in past year. Occasional tinnitus in left ear previous to the present attack. General examination essentially negative. Blood pressure 100/80.

Auditory examination showed hearing loss for low tones of 10 to 15 db. in left ear. Normal tympanic membranes.

No spontaneous nystagmus in upright position.

Positional tests: One week after onset of attack.

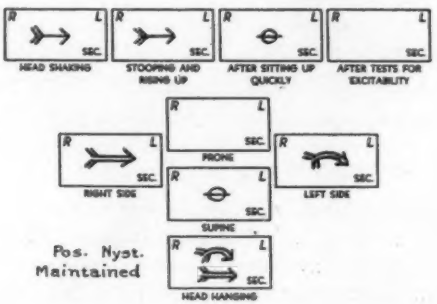

Fig. 2.

Cold caloric tests: Normal responses.

Diagnosis: Vertigo of labyrinthine origin.

Etiology: Uncertain but probably an early Ménière's disease.

No relief obtained from tubal inflation or from intravenous histamine therapy.

Case 2: G. M., female, age 35. Recurring attacks of vertigo for eight months. Mild tinnitus and mild low tone hearing loss in right ear, the severity of which did not seem to be related to the vertigo attacks. Vertigo usually aggravated in certain positions and on movement. History of fall pollen disease, apparently unrelated to vertigo attacks.

General examination essentially negative.

Auditory examination: Low tone nerve type deafness in right ear of mild degree.

No spontaneous nystagmus in upright position.

Positional tests:

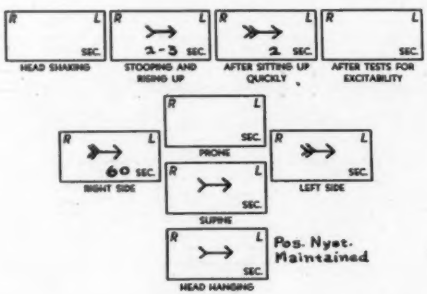

Fig. 3.

Caloric responses normal. Fistula reaction negative.

Diagnosis: Probable Ménière's disease, right ear with postural vertigo and positional nystagmus type II.

Comment: As previously mentioned, a positional nystagmus was rarely seen in Ménière's disease. The validity of the diagnosis in the two illustrated cases could only be borne out by prolonged observation. Case 1 is not known to have had further attacks in a period of about two years, but Case 2 has had several recurrences of a mild nature and the diagnosis appears definitely substantiated.

Case 3: A. H., female, age 28. Sudeck's atrophy of right upper extremity following injury in an automatic wringer nine months earlier. Manipulation and application of splint under ethylene anesthesia on Jan. 26, 1939. Two days later, she complained of severe vertigo on lying on the left side, accompanied by severe left-sided tinnitus. No previous vertigo or auditory symptoms:

Auditory examination: The left ear showed a threshold loss of approximately 30 db., and the right a similar curve with threshold loss of about 20 db. The appearance of tympanic membranes was normal both on Jan. 24, 1939, and Jan. 30, 1939.

No spontaneous nystagmus in upright position.

Positional tests:

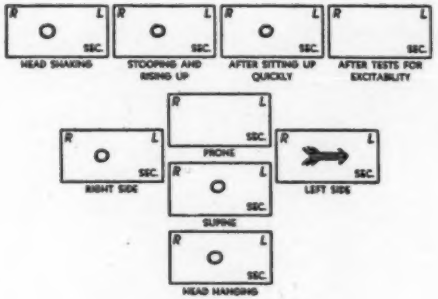

Fig. 4.

Cold caloric responses normal. Fistula reaction negative.

Diagnosis: An acute lesion of the left labyrinth or VIIIth

nerve with mild nerve type deafness, postural vertigo and positional nystagmus type II.

The etiology was indefinite, but related apparently to the operative procedure. A small vascular lesion seems most probable. The postural vertigo persisted unabated for about two weeks, then gradually disappeared.

Case 4: J. G., male, age 26. Acute nonsuppurative otitis media, right ear, several days' duration. Severe vertigo on awakening and turning over to stop the alarm clock. Thereafter, vertigo appeared after a latent period of a few seconds after head movement in any direction, increased to a maximum in a few seconds, then decreased and disappeared. Lying in supine position on a cart in the elevator, the vertigo appeared each time the elevator started or stopped. The latent period then seemed shorter. Direction of nystagmus was constant. Roaring tinnitus in right ear for a period of about two hours out of the 10-hour period of vertigo. Many previous attacks of suppurative and nonsuppurative otitis media. Vertigo on one occasion. Local examination showed subsiding upper respiratory infection and acute nonsuppurative otitis media, right.

Auditory function: Hearing impairment of 10-15 db. for all tones in the right ear and marked bilateral loss at 8,192.

Spontaneous nystagmus: No nystagmus in upright position after being at rest for about one minute.

Positional tests:

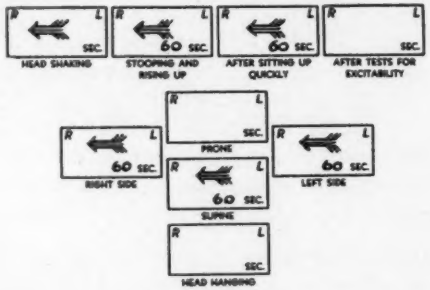

Fig. 5.

Cold caloric response. Normal bilateral.

Fistula reaction negative.

Diagnosis: Acute nonsuppurative otitis media right with postural vertigo and positional nystagmus type II.

Inflation of left ear per catheter — gave no relief.

Inflation of right ear caused reversal of the apparent movement of the environment and of the positional nystagmus.

The postural vertigo persisted for about 10 hours, the characteristics remaining essentially the same, and the hearing loss continued for several days.

Comment: The occurrence of vertigo attacks in nonsuppurative otitis media is well recognized but must be considered as a relatively infrequent occurrence in that condition. The pathogenesis is questionable but in view of the experiments with irritative substances in the middle ear window niches, a mild degree of inflammatory or toxic reaction seems the most likely explanation.

The fact that a sudden change in pressure, produced by inflation of the middle ear, influenced the symptoms but did not abolish them is not easily explained in the absence of a positive fistula sign.

Discussion: In addition to these examples of a positional nystagmus in peripheral ear lesions, it is necessary to mention circumscribed labyrinthitis with a labyrinthine fistula.

In the presence of a fistula a nystagmus may appear in certain head positions and may vary in direction. In such cases, however, the stimulus is usually created by an alteration of pressure at the point of the fistula rather than by the inflammatory reaction. Such cases are not included in the classification of true postural vertigo or positional nystagmus.

The pathogenesis of the positional nystagmus in labyrinthine disease is not yet clear. In some cases the symptom seems

to represent a mild degree of labyrinthine reaction, comparable to a latent spontaneous nystagmus, but in Cases 3 and 4 such an explanation was scarcely tenable.

In Ménière's disease where localized distortions of the membranous labyrinth occur as a result of the hydrops and the function of individual receptor organs may be thereby temporarily affected, a disturbance caused by a change of position seems to have a possible explanation.

The postural character of symptoms in these peripheral lesions would appear to depend primarily on the localization of the disturbance to certain receptors, and secondarily on the degree or severity of the disturbance.

Positional nystagmus is a relatively infrequent occurrence in lesions known to be situated in the peripheral ear. In all such cases so far observed it has conformed essentially to type II, with the direction constant.

POSITIONAL NYSTAGMUS TYPE II IN CENTRAL LESIONS.

Type II positional nystagmus is not confined, however, to peripheral lesions and has been found to be a frequent occurrence particularly in cerebellopontine angle tumors by Nylen[7] and retrolabyrinthine affections by Seiferth.[6] Two cases are briefly described as illustrative of this type in lesions of the central nervous system.

Case 5: P. H., female, age 26. Cerebral concussion following an automobile accident. Moderate vertigo with nausea for four to five days gradually receding. No hearing loss or tinnitus. At the end of one week there was no longer any vertigo while lying in the supine position, but turning on left side produced marked vertigo and nausea. No skull fracture demonstrable.

Auditory function: Normal. No spontaneous nystagmus in supine position.

Positional tests: On left lateral position marked vertigo and nystagmus toward the right. Right lateral position negative.

Cold caloric responses normal.

Diagnosis: Cerebral concussion, postural vertigo, positional nystagmus type II.

Comment: Vertigo may recur at intervals during the months following concussion, but may be only postural. The tests for positional nystagmus are useful during the period when vertigo can be brought on to obtain objective evidence. Either type I or type II may be found.

Case 6: R. B., female, age 26. Complained of severe vertigo whenever she would stoop far forward but not in the upright, supine or lateral positions. No auditory or other specific complaints except general fatigue. The symptom was present for a period of about two months without remissions. Patient complained of general lassitude and fatigue but continued to work. Occasional evening elevation of temperature of about one degree. No previous attack of vertigo.

Neurological, medical and otolaryngologic examinations were otherwise negative. Blood pressure within normal range.

Auditory function normal.

No spontaneous nystagmus in upright position. Pressure over carotid sinuses negative. Fistula tests negative.

Positional tests: No nystagmus in supine, lateral or head-hanging positions. On stooping forward, a vertical nystagmus of moderate intensity occurred regularly. Caloric responses were normal.

Impression: Postural vertigo and positional nystagmus type II, due probably to a central disturbance. Sometimes classed under "pseudo-Ménière's syndrome."

The etiology was indefinite but a toxic factor seemed most probable, although no focus could be demonstrated. The symptoms persisted for a two-month period but have not recurred in four years. A circulatory or vasomotor insufficiency was also considered as a possible explanation.

POSITIONAL NYSTAGMUS TYPE I IN KNOWN DISEASES OF THE CENTRAL NERVOUS SYSTEM.

Case 7: F. G., female, age 37. Complained of headache in right parietal region and above right ear, of three weeks' duration. Vomiting for three weeks. Dizziness for two weeks which was relieved by lying on right side, but appeared on lying on her back or left side and on arising quickly. History of having had a right simple mastectomy nine months previously for removal of cancer. General examination showed healed mastectomy scar. Several firm nodules palpable in right axilla.

Neurological examination gave essentially normal findings except for weak convergence, slight bilateral inaccuracy on finger-nose test and for rapid alternating movements on the left.

Auditory function: Normal for whispered voice and watch tests. No tinnitus.

Spontaneous nystagmus: Absent in the right lateral position which the patient preferred to assume.

Positional tests:

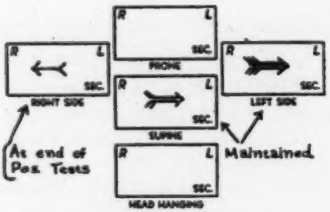

Fig. 6.

Caloric tests normal.

Impression: Probable posterior fossa tumor, metastatic from carcinoma of the breast. Postural vertigo. Positional nystagmus type I. At operation a carcinomatus mass was found and removed from the right cerebellar hemisphere.

Comment: While this case illustrates the early appearance of a postural vertigo and positional nystagmus of type I in a posterior fossa tumor, type II may also occur (Nylen).

Case 8: M. F., female, age 23. Patient had been under observation for the past year in the neurological department with diagnosis of multiple sclerosis. Duration of illness about two years. Referred to otology because of severe vertigo, nausea, and vomiting of nine days' duration. Symptoms present when lying down or turning suddenly. Loss of 10 pounds in nine days since onset of vertigo.

Auditory function: Normal.

No spontaneous nystagmus in upright position.

Positional test:

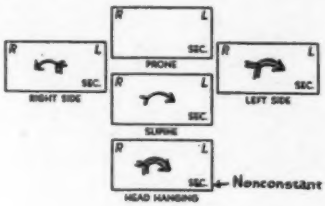

Fig. 7.

Cold caloric responses within normal limits.

Impression: Multiple sclerosis. Postural vertigo and positional nystagmus type I — due to disturbance of central vestibular pathways.

Comment: Observations on known central lesions such as tumors, multiple sclerosis and cerebral concussion agree that a positional nystagmus type I invariably indicates a central location, whereas type II may occur either in a peripheral or a central lesion.

POSITIONAL NYSTAGMUS IN SO-CALLED PSEUDO-MENIERE'S SYNDROME.

Case 9: E. N., female, age 60. Complained of dizziness of three weeks' duration. Vertigo came on after arising in the

morning and gradually became severe with vomiting. Confined to bed for two weeks. Since then, vertigo recurs on lying on either side, on turning the head, or riding in the car. Patient under treatment for the past two years with diagnosis of primary hypertension, retinal and cerebral arteriosclerosis.

Auditory function: No auditory symptoms associated with the vertigo. Examination showed bilateral nerve deafness for tones above 2,048 cycles.

No spontaneous nystagmus.

Pressure over carotid sinuses negative.

Positional tests:

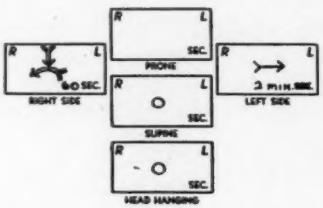

Fig. 8.

Cold caloric responses normal.

Impression: Postural vertigo. Positional nystagmus type I.

Etiology: A small vascular lesion involving central vestibular pathways seemed indicated by the absence of related auditory symptoms, the presence of type I positional nystagmus, hypertension and cerebral arteriosclerosis, and the characteristic onset and course of the vertigo.

Case 10: R. W., male, age 65. Examined two days after a fall during an attack of vertigo. Onset of vertigo occurred while sitting on toilet seat leaning forward, after having arisen from bed and walked to the bathroom. Thereafter, vertigo occurred only in certain postures. Patient was under

observation in hospital with arteriosclerosis. Blood pressure when last recorded before onset of vertigo was 104/66.

No previous attacks of vertigo.

Auditory function: Hearing grossly normal for whispered voice, watch and tuning fork tests. No auditory complaints.

No spontaneous nystagmus in upright position.

Negative response to pressure over carotid sinuses.

Positional tests:

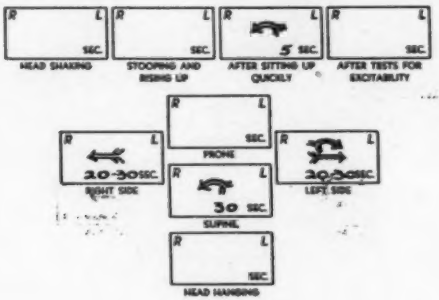

Fig. 9.

During the tests, vertigo was irregular in occurrence and depended upon the rapidity with which the position was altered.

Cold caloric responses normal.

Impression: Postural vertigo, positional nystagmus type I.

Etiology: A circulatory disturbance affecting the central vestibular pathways is suggested by the absence of auditory symptoms and the presence of abnormally low blood pressure, and is further confirmed by the finding of a type I positional nystagmus.

The vertigo gradually disappeared and had not recurred several months later.

Comment: Cases 9 and 10 are described because they are

in the same age period, both with arteriosclerosis and both would usually be classed as "pseudo-Ménière's" syndrome.

A significant distinction can, however, be made between the two. In the case of hypertension, the vertigo required a short space of time to reach a peak, remaining severe for one to two weeks with gradual recession of symptoms during a four-week period. The postural character was evident after the acute stage had passed. Such a course is strongly suggestive of a localized destructive type of lesion, probably vascular, requiring a definite period for compensation.

In the case with a low blood pressure, the vertigo appeared first with the head in a forward position and thereafter could only be reproduced in certain positions. If the position was changed very slowly, the vertigo frequently did not occur, and when it appeared reached a maximum within a few seconds, then faded out within about 30 seconds. Such a course is best explained as a temporary circulatory deficiency of vasomotor origin, affecting the vestibular nuclei or pathways rather than a vascular injury.

The syndrome of vertigo associated with hypotension is not necessarily related to arteriosclerosis since it appears frequently in the third and fourth decades in the absence of demonstrable arteriosclerosis.

Case 11: J. S. Onset of vertigo in the morning after awakening, brought on by turning over in bed or on stooping forward. Slight vertigo while lying in supine position, but severe vertigo and nausea on turning to lateral positions or stooping forward. Symptoms could be avoided by changing position very slowly. Gradual decrease of symptoms over a period of several days. Able to carry on his work after second day.

No auditory symptoms noticed.

History of momentary vertigo and falling on one occasion many years earlier on jumping out of bed to answer the telephone. Occasional mild, momentary dizziness since then. General examination was negative except for a blood pressure

of 88/70, taken at the end of the otological examination. Patient's blood pressure on repeated former observations averaged 110 systolic.

Auditory function: Normal for whispered voice and forks up to 4,096 inclusive. Eustachian tubes inflated on Valsalva's test and deflated on swallowing. No change in vertigo after Politzerization of tubes.

No spontaneous nystagmus.

Pressure over carotid sinuses negative.

Positional tests: On third day of attack.

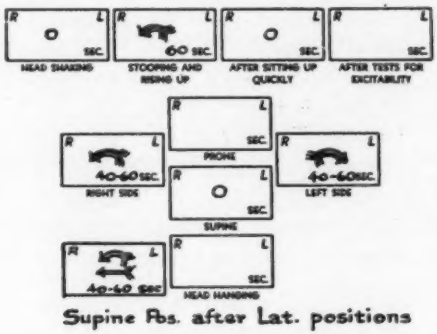

Fig. 10.

Cold caloric responses normal.

Impression: Postural vertigo. Positional nystagmus type I.

Etiology: Circulatory or vasomotor deficiency affecting central vestibular pathways, associated with hypotension.

The vertigo lasted about one week, after which the systolic blood pressure was again 110. There has been no recurrence in the past two years.

Case 12: G. G., female, age 31. Onset of vertigo on awakening three days ago. Vertigo not present in upright position

or walking around, but marked on lying on either side, on her back, stooping over or looking up.

No deafness, tinnitus or sensation of blockage of ears.

History of a similar attack of vertigo, postural in character, six months earlier, lasting four or five days, and a second attack six weeks later. There had also been other very slight attacks. All had been postural in character.

General examination was essentially negative except for blood pressure of 84/56.

Auditory function normal.

No spontaneous nystagmus.

Pressure over carotid sinuses negative.

Positional tests:

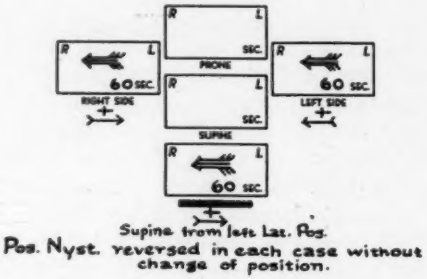

Fig. 11.

Cold caloric responses: Normal. Fistula test negative.

Impression: Postural vertigo. Positional nystagmus type I.

Etiology: Circulatory or vasomotor deficiency affecting central vestibular pathways, associated with hypotension. The period of postural vertigo lasted about six days, the positional tests showing some variation as the symptoms decreased.

Discussion: Cases 11, 12 and 13 are representative of a group characterized by an attack of vertigo which tends to be

postural in type and abnormally low blood pressure, without auditory or other neurologic signs. The attacks may occur in early as well as middle or late adult life. There may be no recurrence, or attacks may recur after one or more years, or within a few weeks. While not associated with any definite systemic disease, other symptoms are common, such as headaches and mild gastrointestinal complaints, fatigue and nervousness which may be related to low blood pressure and poor vasomotor control. Approximately one-half of those patients commonly classed as "pseudo-Ménière's" syndrome appeared to fall into this group.

The other half comprised cases where the vertigo could be ascribed to such causes as hypertension and arteriosclerosis, toxic involvement due to drugs or infection, an occasional case which later proved to be Ménière's disease, and some of indefinite etiology.

DISCUSSION.

Observations that have been made in known diseases of the central nervous system, such as concussion, encephalitis, tumors and multiple sclerosis, illustrated by Cases 5, 6, 7 and 8, indicate that a positional nystagmus can be demonstrated more frequently than a spontaneous nystagmus in the upright position in such conditions. A positional nystagmus which is irregular in its direction or which changes direction with changes of position (type I) appears to be a reliable indication of a centrally located lesion, whereas a positional nystagmus with constant direction (type II) may occur in either a peripheral or a central lesion.

In those cases of vertigo without auditory disturbances or central neurologic signs (pseudo-Ménière's syndrome), a positional nystagmus has proven to be an almost constant feature. The postural character of the vertigo may be evident throughout or may appear as the more severe stage of the attack passes off. The reproduction of the syndrome on the positional tests provides objective evidence which might not be obtainable either on examination of vestibular function which is usually normal, or for spontaneous nystagmus in the

ordinary upright position since the latter sign may be absent.

On the basis of the classification established in known lesions, the positional nystagmus in this group has been predominantly of type I, indicating a probable central origin. Type II has been rare, although when present would not contraindicate a central lesion.

By the correlation of the positional nystagmus with the history and general examination of the patient, it is possible to differentiate certain groups within this so-called "pseudo-Ménière's" group.

Approximately one-half consisted of cases that could be ascribed to such factors as hypertension and arteriosclerosis, toxic involvement by drugs or infection, and some of indefinite etiology. It is probable also that a progressive disease of the central nervous system might produce such vertigo as the first symptom.

The other half, illustrated by Cases 10, 11 and 12, fell into the group characterized by an abnormally low or labile type of blood pressure, sometimes with frequent headaches, mild gastrointestinal disturbances, and signs of poor vasomotor control. The attacks have sometimes not recurred in a period of several years, while some have had one recurrence and some several attacks in from one to 10 years.

The fact that Ménière's disease, which is an inner ear disease associated with labyrinthine hydrops, sometimes also develops in cases with hypotension, suggests an etiological relationship. There are, however, characteristic differences in that Ménière's disease is less common under 40 years of age and frequently appears also in later decades in the presence of high blood pressure and, although an attack of vertigo may be the earliest symptom, the auditory disturbances are the most constant feature of the disease.

The reason for the postural character of the vertigo remains a matter for speculation. In the case of the peripheral labyrinthine lesion, the postural character may appear only in the milder stages of the reaction or the vertigo may be postu-

ral throughout, although of severe intensity. A localization of the lesion to certain receptors, probably the utricle, seems a possible explanation in the early stage of inflammatory disease or in labyrinthine hydrops with distortion of membranous structures. The postural feature of the vertigo which occurs in a central lesion may also be apparent only in the receding stage of a severe attack or may be characteristic from the onset. In the case of inflammatory and progressive degenerative diseases and severe grades of hypotension, the appearance of the symptom seems to have its most probable explanation on the basis of a local circulatory or vasomotor insufficiency. The fact that the vertigo may usually be avoided by changing position slowly seems to support this view.

TREATMENT.

The treatment in a case presenting pseudo-Ménière's syndrome depends basically upon the etiology. The vertigo is only a symptom which may be alleviated by rest and mild sedation, but beyond that the treatment is directed at the etiologic condition. Surgical interference is not indicated for relief of the vertigo.

SUMMARY.

1. Postural vertigo and its accompanying positional nystagmus is the most common type of vestibular disturbance in diseases of the central nervous system. It occurs less frequently in peripheral ear diseases. The syndrome can be reproduced on routine positional tests.

2. Observations in known lesions, central and peripheral, indicate that two types of positional nystagmus may be differentiated. Type I, which is irregular in direction or changes direction with alteration of position, is found only in diseases involving the central vestibular pathways, while type II, with constant direction, may occur in either central or peripheral diseases.

3. Attacks of vertigo of the pseudo-Ménière's type, in which auditory disturbances and central neurologic signs are absent,

have been found to exhibit a postural character as a predominant feature.

4. In a series of over 30 cases of the pseudo-Ménière's type with positional nystagmus at the time of examination, type I was found in all but one. This would appear to be strong evidence of a central location of the disturbance.

5. It is possible on the basis of associated systemic signs to differentiate several groups within the general pseudo-Ménière's group, such as hypertension and arteriosclerosis, toxic involvement due to drugs or infection and hypotension, while a small number remain doubtful as etiology. Approximately one-half of this series could be included in the hypotension group on the basis of low blood pressure and evidences of vasomotor insufficiency.

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THE OCCURRENCE OF SPONTANEOUS CEREBROSPINAL RHINORRHEA IN THE LITERATURE, THE EXPERIENCE OF THE WRITER, AND OTHER DIPLOMATES OF THE AMERICAN BOARDS OF OTOLARYNGOLOGY AND NEUROSURGEONS.*

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Cerebrospinal rhinorrhea is the escape of spinal fluid through the nose as the result of trauma, intracranial disease or unknown etiology.

HISTORICAL.

In 1826, Charles Miller, of Edinburg, Scotland, was the first to report and correctly recognize the condition. His paper was brilliant and was read before the Medico-Chirurical Surgical Society of that city.

Willis, in 1676, and Morgagni, in 1762, described fluid escaping from the nose, but neither recognized its true nature.

Tillaux, in 1877, proved his case to be cerebrospinal rhinorrhea, by chemical examination.

Leber reported a case in 1883, which he attributed to internal hydrocephalus.

In 1899, Sir St. Clair Thomson published his monumental work, "The Cerebrospinal Fluid, Its Spontaneous Escape Through the Nose," in which he reports 21 cases, nine definite ones and 12 probable ones. Every rhinologist should read his monograph.

Johnson,[1] in 1926, reported a case and also summarized the literature to that time.

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Locke,[2] in 1929, reviewed the literature and reported autopsies on 14 cases under the heading of pathology.

Johnson,[3] in 1929, reported a case of cerebrospinal rhinorrhea in a three-year-old child due to internal hydrocephalus.

Friedman reports two cases, one due to pituitary tumor and one due to hemorrhage in internal capsule.

Learmouth reported granulation tissue noted on cribriform plate at time of operation.

Wurster,[4] in 1937, reported a case.

Cerebrospinal rhinorrhea is but briefly considered in textbooks on rhinology. This condition has been regarded as extremely rare.

In the preparation of this paper, a questionnaire was mailed to each otolaryngologist and neurosurgeon certified by his respective board. There were slightly more than 3,000 questionnaires mailed, the names having been taken from the 1942 edition of *Medical Specialists*. Replies were received from 1,598 of these specialists.

It is almost impossible to separate traumatic and spontaneous cases. Judging from the answers to the questionnaires, however, I am struck by the fact that the condition occurs more frequently than we formerly believed, its recognition depending on the accuracy of the observer.

ETIOLOGY.

Cairnes[5] lists the causes of cerebrospinal rhinorrhea:

1. Those occurring during acute stages of a head injury.

2. Those that occur as delayed complications of a head injury.

3. Those produced during operations on the nasal accessory sinuses or the cranium.

4. In spontaneous cases of cerebrospinal rhinorrhea, it is

necessary to investigate the frontal, ethmoid and sphenoid sinuses.

Locke[2] classes all cases of spontaneous cerebrospinal rhinorrhea as "tumor suspect" and every possible examination must be made to rule out neoplasms in the skull cavity. Increased intracranial pressure or some obstruction to the flow of cerebrospinal fluid is usually noted.

5. Friedberg and Galloway[6] make the interesting observation that when the middle turbinate is not disturbed in intranasal surgery, no cerebrospinal rhinorrhea results.

6. Many cases are the result of old injuries to which the patient apparently attached no significance. Tumors of the pituitary, luetic, lesions, and osteomas of the frontal and ethmoid sinuses, are reported as common causes.

In cases of internal hydrocephalus, the cerebrospinal rhinorrhea manifested itself subsequent to the closure of the fontaneles and suture lines.

Congenital defects in the cribriform plate — Cairnes[5] reports two cases.

1. Nasal cephalocele.

2. Cerebellar astrocytoma confirmed by operation and Roentgen findings. Small meningoceles may extend through the cribriform plate in the region of the olfactory nerve exits and appear in the nasal cavities as polypi.

Loftus[7] states that defects occur in the craniopharyngeal wall, this embryonic canal being forced open by increased pressure of the cerebrospinal fluid.

Another theory suggests that lymph passes from the arachnoidal spaces opening on the nasal mucous membrane. Some claim that the fluid escapes along the perineural sheaths of the olfactory nerves.

Cases also occur secondary to surgery on the ethmoid, frontal and sphenoid sinuses, as well as after submucous resections.

SYMPTOMS.

A variety of symptoms may precede the onset of cerebrospinal rhinorrhea: headaches, dizziness, eye symptoms, failing vision, optic neuritis, scotomas, bitemporal, hemianopsia, etc.

Convulsions and paralysis may occur prior to or during an attack.

Frequently the onset is ushered in by the patient noting a clear, colorless, odorless, limpid fluid escaping from one or both sides of the nose. It may be constant or intermittent. Some say it has a slightly salty taste. It may be preceded by sneezing. There may be no particular nasal discomfort. The fluid is free of sediment and mucin. It does not excoriate the nasal vestibules or the lips. There are no conjunctival symptoms as seen in vasomotor rhinitis or other allied allergic conditions in which sneezing occurs paroxysmally and the discharge is irritating to the skin.

The cerebrospinal fluid does not stain linen nor are handkerchiefs stiff after drying. In cerebrospinal rhinorrhea, the condition persists even during sleep, the patients usually swallow the fluid and one may note a gurgling sound in the throat.

The condition is also more noticeable when the patient leans forward. The cerebrospinal fluid may gush out almost in a steady stream; usually, however, it occurs in a constant drip and is readily collected in a sterile receptacle. The amount varies from a few cc. to 2,000 cc. in a 24-hour period.

Sir St. Clair Thomson suggests the following criteria for positive identification

1. The fluid is perfectly transparent like water, and contains no sediment.

2. It is faintly alkaline in reaction, and either tasteless or slightly salty.

3. The specific gravity is between 1.005 and 1.010.

4. It is not viscid and gives no precipitate (mucin) on adding acetic acid.

5. On boiling, there is not more than a trace of coagulum of serum globulin and serum albumin.

6. Cold nitric acid gives a precipitate which disappears on heating, and separates again on cooling.

7. Saturation with magnesium sulfate should give a precipitate. Saturation with sodium chloride should also produce a precipitate. Ammonium sulfate should be tried if the above salts fail.

8. The liquid should give a pink or rosebud color with a trace of copper sulfate and excess of caustic potash.

9. When boiling with Fehling's solution, there should be a reduction of the copper.

10. The reducing substance may be obtained by evaporating to dryness an alcoholic extract of the fluid. It is then found in the form of needle-like crystals.

11. The aqueous solution of this residue does not ferment with yeast.

The cerebrospinal fluid should be examined promptly, this being especially necessary to procure an accurate determination of its sugar content. On standing, certain chemical changes take place, after which examinations are inaccurate.

Nasal examination is usually negative. Some authors state that patients note a "damp feeling" in the nose prior to the onset of the rhinorrhea. It may drip for a few days, cease for a considerable time and then recur.

CHARACTERISTICS OF THE FLUID.

The fluid is clear, colorless, odorless and its specific gravity is 1.005 to 1.010. It is usually sterile; neutral or faintly alkaline to litmus; contains no sediments; mucin and albumin are absent or present in minute quantities; cell count is from one to 20, shows the presence of copper reducing substance (glu-

cose). The amount of solids is about 1 gm. per 100 cc. The sugar varies from 0.032 to 0.148 gm. per 100 cc.

PATHOLOGY.

Lederer[8] mentions three possible routes of escape of cerebrospinal fluid into the nose due to neoplasm of the central nervous system, namely:

1. Communications from the floor of the anterior horn of the dilated lateral ventricle, through the eroded cribriform plate into the nose.

2. Openings from a persistent lumen in the olfactory bulb, through the cribriform plate into the nose.

3. An opening from the nasal subarachnoid cistern through the cribriform plate into the nose.

The cerebrospinal fluid is separated from the nasal sinuses and the nares from within out by the arachnoid, dura, thin bony partition and mucous membrane of the nasal sinuses and the nose. Thus with this brief review of anatomy in mind, it is readily seen that fractures, trauma, tumors and infections can readily produce changes that lead to the escape of the cerebrospinal fluid. The change may be pressure atrophy of the dura and bone. Shea[9] describes such a case following trauma.

Eagleton[10] explains the persistence of a craniosinal fistula which results from fistulous traumatic or olfactory perineural tract by the ingrowth of low-grade mucous membrane combined with proliferation of the arachnoid when the two membranes are approximated. This membranous growth in flat bones prevents bony or fibrous union that would normally take place. The persistent flow of cerebrospinal fluid soon converts this tract into a permanent endothelial lined channel. Continued abnormal brain pressure causes more bone absorption in the cribriform plate, which conducts the perineural arachnoid prolongations from the olfactory bulb. Moreover, the arachnoid lake and the nasal mucous membrane become continuous where the traumatic or perineural tract

enters the chiasmal cistern. This may form an intercerebral cyst which spreads through the sylvian fissure, meshes to the cortex and nasal cisternia when the defect is not properly treated. Thus we have explained the formation of the cranionasal and craniosinal fistulas.

Patients with cerebrospinal rhinorrhea should never strain, cough or sneeze violently, or blow the nose. Air in the brain or pneumatocele may result in sudden loss of consciousness, convulsions, etc. When the patient is erect and the fluid is escaping, the intracranial pressure is lower, and thus virulent organisms may enter and produce a fatal meningitis.

In this paper we are dealing primarily with spontaneous cases, but it is practically impossible not to discuss the connection with trauma. Undoubtedly, the steps in the adequate treatment of spontaneous cerebrospinal rhinorrhea have been wonderfully helped by advances made in handing traumatic cases.

Adson's[11] excellent article gives the causation of cerebrospinal rhinorrhea as skull fractures through the posterior wall of the frontal sinus or the cribriform plate of the ethmoid bone with accompanying lacerations of the dura and arachnoid. Many of these cases develop a watery, bloody discharge that stops spontaneously. There is apparently some controversy among neurosurgeons as how best to handle these cases. The advent of the sulfa drugs enters the picture. Many cases probably are never recognized. Those that persist are the result of failure of fracture to heal, absorption of bone, etc., or where the dura and arachnoid have been caught between the bone fragments.

Adson feels that probably cerebrospinal rhinorrhea occurs spontaneously when the arachnoid envelope extends along olfactory nerve fibres through the cribriform plate of the ethmoid bone, thus rupturing the thinned arachnoid and nasal mucous membrane.

Wauregg thinks sneezing may precipitate such a condition. Some men attribute it to attacks of influenza because influenza is said to raise intracranial pressure, and the leak devel-

ops. In any case, where such a thinning out is present, any sudden increase in intracranial pressure would naturally precipitate the condition.

Pituitary tumors are a common cause, as will be shown in the review of the questionnaires previously mentioned.

Adson[11] reports a case of pituitary tumor eroding through the floor of the sella turcica and leakage of cerebrospinal fluid into sphenoidal sinuses. He cites another case of osteoma of the ethmoid and orbit.

Several men mentioned cerebrospinal rhinorrhea following the removal of nasal polypi. These polyps are probably meningoceles.

Som and Kramer[12] state that the cerebrospinal fluid escapes from the nose as the result of:

1. Trauma.

2. Neoplastic invasion of the skull.

3. Infection.

4. Increased intracranial pressure at times called spontaneous rhinorrhea.

5. Idiopathic.

a. We have previously described.

b. Neoplastic invasion of the skull or dura. The tumor may originate in the bone proper, *viz.*, frontal osteoma; or the bone may be invaded as in pituitary adenoma or craniopharyngioma.

c. Infection of the paranasal sinuses may produce a suppurative necrosis of the floor of the anterior fossa and result in a fistulous tract.

d. Cerebrospinal rhinorrhea may be associated with a long-standing increased intracranial pressure as a result of brain tumor on internal hydrocephalus. Pituitary neoplasm or the posterior fossa frequently produce this type of internal hydrocephalus.

e. Pure spontaneous rhinorrhea, as described by Sir St. Clair Thomson, where rest in bed and spinal puncture effect a cure, is hard to explain due to the lack of autopsy findings, etc.

Ballon and Ballon[13] report pin point openings in the dura and cribriform plate.

Livini[14] regards these as congenital dehiscences. Cairns[5] reports two cases of brain tumor in which he found a circular deficiency of the cribriform by X-ray examinations. Shea[9] reports a case of marked hydrocephalus and cerebrospinal rhinorrhea where there was seen at postmortem an erosion of the roof of the right ethmoidal labyrinth resulting in a fistula between the right anterior fossa and an ethmoidal cell containing hemorrhagic polypi.

Som and Kramer[12] report autopsy findings on a female, age 44, suffering from hypertension of many years' standing. Five years previously, severe headaches had been relieved by lumbar puncture. For the past three years, severe headaches were relieved by intermittent escape of clear fluid from nose.

Patient died of meningitis. Autopsy showed a large pituitary neoplasm. Coronal serial sections of the paranasal sinuses which were removed *en bloc* were studied. A dural polyp and nerve contents had herniated through the cribriform perforation and a fistulous tract formed. This was the probable portal of entry of the meningitis, and, likewise, acted as a valve to regulate the escape of cerebrospinal fluid, thus controlling the intracranial pressure. Several authors mention the decompressing effect of the cerebrospinal fluid. Som and Kramer[12] report a second case admitted with upper respiratory symptoms with bilateral sphenoethmoiditis. Left sphenoethmoidectomy was done, but the right sphenoid was not entered due to patient's condition. Autopsy showed a submucosal abscess in the posterior wall of the right sphenoid sinus. Adjacent to this was acute osteomyelitis of bony wall of right sphenoidal sinus. The bone was eroded with a fistulous tract overlying the dura. This resulted in a fatal meningitis.

Shea[9] reports autopsies of three cases:

1. Basal skull fracture with dura tear.

2. Pituitary tumor with the sella turcica, and erosion of the clinoid processes and a defect in floor of sella turcica opening into sphenoid sinus.

3. Erosion of right ethmoid labyrinth following arachnoiditis causing internal hydrocephalus resulting in pressure and thinning of dura and bone.

Berger[15] reports a case of cerebrospinal rhinorrhea in a female, age 31, first seen in April, 1932, with cerebrospinal rhinorrhea associated with craniopharyngioma.

Wessells[16] reports a case in which a fistula in the floor of the anterior fossa extended through the left cribriform plate. Dr. Sachs found this condition at time of surgery.

Titche,[17] June, 1941, reports a case of hyperostosis frontalis interna diagnosed by Roentgen examinations which may have caused stretching and tearing of the dura over the cribriform plate. Anatomically, we know that the dura is thin here and extremely adherent.

Perot[18] reports a case of tumor of the frontal lobe associated with cerebrospinal rhinorrhea.

Britt[19] presents two cases of rhinorrhea and neoplasms of the central nervous system.

Smith and Walter,[20] in 1931, reported a case of cerebrospinal rhinorrhea with cyst of the pituitary body found at the operation. The cyst was opened and fulgurated. No fistulous tract was noted, although X-rays revealed absorption and thinning of the floor of the sella turcica. This patient recovered and is now in good health.

Wurster,[4] in 1932, reported a case of spontaneous cerebrospinal rhinorrhea which had undergone numerous intranasal operations, including two submucous resections, turbinectomy, tampons, violet ray, etc., which spontaneously ceased after several years. Patient is now in excellent health.

Bulson and Bulson[21] saw a female, age 40, in February, 1921, for a routine refraction. Upon returning on Sept. 12, 1922, she stated that two months previously she had had a nasal operation, both turbinates from each side having been removed. This was followed by a profuse watery discharge. She refused hospitalization and further examinations. This patient was kept under observation for six years and the discharge gradually lessened, becoming periodical and at times ceasing.

This case is especially interesting since there was a large pulsating mass in the region of the posterior ethmoidal cells on the right side. Granulations apparently were slow but successful, since no meningitis resulted. No intranasal manipulations were done. Another remarkable feature was that on one occasion 32 ounces of fluid were reported lost in 24 hours.

Campbell[22] reports eight cases following intranasal surgery. Three died of meningitis. Five recovered within a few days to a few weeks. This was prior to the days of sulfa drugs.

Plunkett and Lendrum[23] report a case of spontaneous pneumocephalus and cerebrospinal rhinorrhea, a male, age 54, with a diagnosis of combined interstitial parenchymatous syphilis of the central nervous system. After much treatment, he was undergoing spinal puncture; no spinal fluid ran out of the needle, but a hissing sound was noted, due to the air entering the needle. The patient was X-rayed and showed air in the ventricles and subarachnoid spaces. The patient died suddenly and autopsy showed a circular hole with smooth edges in the base of the skull just posterior to the cribriform plate of the ethmoid bone. The posterior ethmoid, sphenoid and medial wall of the left antrum were absent, thus throwing these sinuses into one chamber. No cause for the bone lesion could be found. In passing, it is interesting to note that these authors reviewed the literature and found 99 other case reports of spontaneous pneumocephalus.

T. O. Graham,[24] of Dublin, reports a case of cerebrospinal rhinorrhea in a female, age 20, who showed Roentgen changes.

The anterior and posterior clinoid processes had almost disappeared, the pituitary fossa was practically destroyed; and the sphenoid sinuses were all but obliterated. Operation revealed wider openings than normal in the cribriform plate. These were closed with Horsley's bone wax. The patient had no subsequent rhinorrhea, but became fat and developed headaches, which were undoubtedly due to changes in the pituitary body.

Donnelly[25] reports two cases: one spontaneous and the other traumatic. Spontaneous case, female, age 60. Following an attack of influenza, the patient noticed a watery discharge from right side of nose. Nasal examinations showed fluid escaping from the ostium of the right sphenoidal sinus. Roentgen examinations showed a thinning of dorsum sella.

Traumatic case, male, age 57, died from meningitis, following right ethmoidectomy. During the operation, a large polypoid mass was bitten into and cerebrospinal fluid escaped. Autopsy revealed a ragged mass in the right ethmoidal roof.

Sir James Barrett[26] reports spontaneous cerebrospinal rhinorrhea in a patient having bilateral acoustic neuromas.

Bromberg[27] reports a case, male, age 31, with cerebrospinal rhinorrhea with pneumocephalus secondary to skull fracture, involving the frontal and parietal bones on the right side extending over the supraorbital ridge; there was also a fracture of the right nasal bone. X-rays revealed the pneumatocele. By placing patient in a horizontal position, with aid of sandbags, recovery took place.

Rawbothan,[28] in *Acute Injuries of the Head*, discusses delayed cerebrospinal rhinorrhea that occurs months or even years after injury. He attributes the pathology to pull as the result of the cicatrization of the healing processes on a normal fistula which has passed unnoticed and then widened. He also described a condition of osteitis desiccans, which may result from ischemia due to dural tears which interrupt the nutrient blood supply of the bone. The dura and nasal mucous covering the bone become involved in a necrotic process and a fistula results.

Gibbs[29] reports a case in which the only findings were a generalized arteriosclerosis.

The most exact pathological knowledge that I can find in the literature regarding spontaneous cerebrospinal rhinorrhea is the 14 cases of authenticated autopsies reported by Locke[2] which follow:

Obstruction to the flow of cerebrospinal fluid or increased intracranial pressure are unquestionably the two factors that produce a fistula between the anterior cranial fossa and the nose.

It is most probable that increased intracranial pressure causes atrophy of the cribriform plate. The arachnoid membrane and subarachnoid space continue along the olfactory nerves that pass through the dura.

Locke[2] described how celloidon injected under pressure in the subarachnoid space of a dog leaked into the nose. This did not occur in the human cadaver.

When the cribriform plate is atrophied and the normal openings of the dura and arachnoid are enlarged by intracranial pressure, only the nasal mucous membrane remains between the cerebrospinal fluid and the nasal cavity. This nasal mucous membrane is not able to withstand much pressure. It gives way and cerebrospinal rhinorrhea results.

Locke[2] likewise makes the pertinent statement "that probably we do not thoroughly understand the origin and circulation of the cerebrospinal fluid."

G. S. Baker and A. R. McLean[30] discuss cerebrospinal rhinorrhea associated with pneumocephaly, pneumococcic meningitis and abscess of the brain. The case was first seen in July, 1941, with a history of meningitis in 1936 with recovery. In 1940, the patient noted weakness in the right arm and leg and a discharge of clear fluid from the left side of the nose. Headaches, dizziness and diplopia occurred two weeks prior to visit to clinic. X-ray studies showed pneumocephaly and osteoma in left frontal and ethmoid region.

Transfrontal craniotomy revealed an osteoma extending to left sphenoid ridge and a muscle graft was placed over the opening in left frontal sinus.

Adson[11] reports a case of a teacher, female, age 34, who was first seen in 1940. She had had cerebrospinal rhinorrhea at ages 12, 22 and 33, and previous head injuries to which she attached no importance. Nine months before consulting Dr. Adson, she noticed, during conversation, that a clear liquid came from the right side of her nose. X-rays were negative. Sulpha drugs were administered prior to surgery. Transfrontal craniotomy was done and a fistulous tract was observed through cribriform plate on the right side. Horsley's bone wax was molded into the olfactory grooves of cribriform plate, and temporal muscle graft was placed thereon. Sulfa drugs were advantageously used. The patient recovered, although anosmia resulted.

Higbee[21] reports that while doing a radical external frontal operation, the dura was accidentally torn and surgery was stopped. The patient was put in a special frame in exactly the same position as when the surgery was being done, and after a few days recovery took place.

Livini[14] reports a case, a Hindu, age 26, having spontaneous cerebrospinal rhinorrhea, who had sustained a head injury 16 days previously. He suddenly sneezed and cerebrospinal fluid began to flow spontaneously. X-rays and neurological examinations were negative. Spontaneous recovery occurred after six weeks of bed rest.

DIAGNOSIS.

The diagnosis of cerebrospinal rhinorrhea is not too difficult if one will keep the condition in mind.

Every patient should have a complete physical, ophthalmological, neurological, serological, Roentgenological (orbit, skull and nasal accessory sinuses) examination, and any other laboratory examination that might help.

Cairns[5] believes that well taken X-rays of the cribriform

areas help, and feels that tomographic studies eventually will aid materially.

A careful history is essential to differentiate between spontaneous and traumatic cases, special attention being given to history of brain and sinus operations and injuries. Nasal examinations must be painstaking and include all diagnostic procedures. One must be very particular, however, in any unnecessary intranasal manipulation.

The various allergic conditions must be differentiated. In hay fever, etc., paroxysms of sneezing, watery eyes and reddened nasal vestibules are noted. Handkerchiefs are stiff when they dry due to the presence of mucin. In cerebrospinal fluid, linens are soft and pliable. Cerebrospinal rhinorrhea may be bilateral, but is usually unilateral and does not cease during sleep. The fluid usually comes out in drops. The amount varies from a few cc. to one or more ounces per hour. Cerebrospinal fluid will reduce Fehling's solution. It should be examined promptly, because on standing, chemical changes take place.

Compressing the jugular veins will increase the flow of cerebrospinal fluid.

One may inject fluorescein in the spinal canal and discoloration appears in the cerebrospinal fluid. Fox[32] injected a solution of 0.6 per cent indigo carmine in the spinal canal, which resulted in a bluish discoloration in the nasal drippings within 15 minutes and lasted five hours.

Attempts have been made by using cotton pledglets impregnated with fluorescein to locate the draining area in the nose, which is usually between the septum and the middle turbinate.

Dandy[33] cites an exceptional case where cerebrospinal rhinorrhea developed following removal of an acoustic nerve tumor in a male, age 48. The cerebrospinal fluid drained through the nose, escaping through the mastoid bone, middle ear and Eustachian tube. Hence all cerebrospinal rhinorrhea is not due to a fistula into the frontal or the ethmoid sinus.

Operative attempts were unsuccessful, the case terminating in a fatal meningitis.

One should always remember the relationship between headaches and the escape of cerebrospinal fluid. Frequently headaches are relieved temporarily. Without treatment the cerebrospinal rhinorrhea ceases and then the headaches return. One must bear in mind the previously mentioned possibility of brain tumors. The flow of cerebrospinal fluid acts thus in a decompressor manner.

One author reports cerebrospinal fluid draining in the middle meatus. This was due to the craniosinus fistula draining into the frontal sinus and then through the nasofrontal duct.

PROGNOSIS.

Cerebrospinal rhinorrhea is a serious menace to health. Spontaneous recoveries have been reported.

The danger of an ascending infection is ever present. Many cases end in a fatal meningitis; hence, intranasal manipulations should be restricted to a minimum. Formerly with surgery, the fatalities were about 30 per cent, but with improved techniques, the sulfonamides, and penicillin, this has been reduced.

TREATMENT.

Formerly, patients were given no treatment, but with the advent of neurological surgery, definite progress has been made. All neurosurgeons see many traumatic cases.

Many drugs have been used but none other than sulfonamides appears to have any definite value.

The restriction of fluids and maintenance of a semi-recumbent positions seem to have produced good results in a few cases.

Formerly, spinal taps were frequently advocated, but no definite help resulted.

Roentgen therapy, too, has its advocates, but no definite proof has been forthcoming as to the value of this treatment.

Patients with cerebrospinal rhinorrhea should, if possible, avoid upper respiratory infections and extreme fatigue. The constant menace of meningitis must not be forgotten.

Every possible attempt should be made to exclude intracranial neoplasms. Naturally, when present, their removal is necessary before any attempt is made at closing the cranionasal fistula.

Where no proven intracranial pathology can be demonstrated, the judicious use of 20 per cent solution of silver nitrate has resulted in cures; however, this applies to only a few cases.

Here again, the history helps in cases of late and recurrent cerebrospinal rhinorrhea as pointed out by Gurdjian and Webster.[34] Delay in closing the fistula has resulted in brain abscess, meningitis and death, in spite of chemotherapy.

Undoubtedly, cases of trauma are seen where cerebrospinal rhinorrhea goes unrecognized because it ceases spontaneously; hence, considerable controversy exists as to whether acute traumatic cases should be treated immediately or whether one should wait in the hope that cerebrospinal rhinorrhea might cease spontaneously. If there is a recurrence within a few weeks or months, the fistula should be promptly repaired.

The two cases that I am reporting were referred to Dr. Coleman, who repaired the fistulas by transfrontal approach with muscle implants. Since the histories and physical examinations found by Dr. Coleman coincide in detail with mine, I shall only report his findings.

The following methods have been used as cited by Gurdjian and Webster.[34]

Both intradural and extradural approaches are used. The various surgical techniques will be merely mentioned and not fully discussed. A fascial transplant may be used. Fascia lata, temporal fascia, pericranial fascia, osteoperiosteal trans-

plants from the tibia, cautery of the fistula, plugging with muscle transplant and Luken's animal membrane have been used. Fascia lata has been used intradurally without suturing.

Adson[11] uses a bifrontal osteoplastic craniotomy approach in order to secure adequate exposure to identify better the fistulous tract. The dura is more easily elevated along the cribriform plate and the tension of the dura is much less.

German[35] uses the intradural method. A flap from the crista galli and falx is utilized. It serves to repair the dural hole. He feels that with adequate chemotherapy, the hazard of meningitis is reduced at the site of the fistulous tract. He reports five successful cases. Anosmia naturally results on the side where the fistula is repaired. In Adson's method, it is necessary to sacrifice both of the olfactory nerves.

COMMENTS.

The evolution of therapy in cases of cerebrospinal rhinorrhea is a fascinating study The original doctrine advocated absolutely no treatment and patients were left strictly alone. With advances in surgery, particularly neurosurgery, the problem was attacked along more rational lines.

Formerly, intranasal medication was absolutely forbidden. Today, cases are reported cured by the application of drugs intranasally; that is, where no intracranial neoplastic pathology exists.

Neurosurgeons report brilliant results both with intradural and extradural methods of approach.

The mortality, at least all I could be sure of, was 30 per cent from an operative standpoint. Surgery together with chemotherapy surely has and will further simplify matters.

Who knows what the future holds? My firm conviction is that spontaneous cerebrospinal rhinorrhea is amenable to cure when properly studied and correctly handled.

TABULATION OF SPONTANEOUS CASES OF CEREBROSPINAL RHINORRHEA AS RESULT OF QUESTIONNAIRES.

Although no information was requested about traumatic cases and cases following intranasal or intracranial surgery, it is interesting to note in passing that many men reported cases following nasal skull fractures, fractures involving frontal and ethmoidal sinuses, gun-shot wounds, septal operations, surgery involving the frontal, ethmoidal and sphenoidal sinuses, and the removal of nasal polyps. They were handled both conservatively and by surgery. Several cases showed marked improvement with the combined use of the sulfa drugs and penicillin.

The following compilation was made from the cases reported as spontaneous cerebrospinal rhinorrhea and will include a brief summary of each case:

CONGENITAL CASES.

1. List. Female, age 35, congenital defect posterior wall frontal sinus, frontal osteoplastic craniotomy, plastic repair. Muscle placed in bony defect, excellent result.

2. Davidoff. Three cases.

a. Female, age 13. Hemangioma on face, treated with radium. Congenital defect, cerebrospinal rhinorrhea began at age of three years and lasted 10 years. Left-sided rhinorrhea recurred several months after repair. Parents delayed surgery, and the child developed meningitis. Treated with sulfanilamide. Recovered. Second repair three months afterward. No recurrence of rhinorrhea for three years. Results excellent.

b. Female, age 52. Four years before onset of rhinorrhea, patient had left-sided intranasal frontal sinus operation; this may have been a factor. Craniotomy; plastic repair. Results excellent.

c. Male, age 45. Low mentality. Complained of headaches. Convolutional atrophy in his skull and other signs of oxyceph-

aly. Right-sided drip for five weeks before consulting physician. Craniotomy; plastic repair. Results excellent.

3. Furstenburg. Female, age 35. Discharge of fluid from nose. A meningeal encephalocele extended into the nose. Hair line incision, removal of the encephalocele and fascia lata placed over the perforation in the cribriform plate. Excellent results.

4. Ehols. Two cases in young adult females, precipitated by sneezing. Both cured by osteoplastic craniotomy. The extradural procedure was used in each case.

5. Gidale. Reports two cases: female, age 48, and male, age 56, which he contributed to congenital dehiscence in cribriform plate. Absolute bed rest. Recovery.

6. Fitzbugh. Child, age two. Congenital dehiscence. Meningitis. Death.

7. Burman. Female, age 14. Congenital defect. Lost sight of.

8. Marshall. Female, age 12. Constant nasal discharge. No treatment. Patient disappeared.

9. Snapp. Male, three months old. Left-sided rhinorrhea. Patient moved away. Child not seen again.

10. Robinson. Female, age 40. Rhinorrhea present since infancy. No treatment.

CASES DUE TO SYPHILIS.

1. Creasy. Female, age 46. Positive Wassermann. Antiluetic treatment. Meningitis; death.

2. Sanderson. Female, age 50. Necrotic bone and membrane in ethmoid area caused by syphilis. Treatment. Results good.

3. Rich. Age 46. (Sex not given.) Clinic case. Sulfathiazole and antisyphilitic treatment. Good results.

4. Harris. Clinic case, female, age 50. Rhinorrhea. Posi-

tive Wassermann. Treatment. "Lost sight of after two years."

CASES DUE TO INFLUENZA.

1. Hart. Female between 40 and 50 years. Unilateral rhinorrhea. Diagnosis made at Duke University. Two spinal punctures and administration of sulfadiazine. Recovery.

2. Greene. Female, age 51. Influenza in February, 1944. Rhinorrhea since. Nothing locally in nose helped. Sudden cessation of flow following headaches.

TUBERCULOSIS.

1. Reeves. Male, age nine. Increased intracranial pressure presumed due to tuberculoma, rhinorrhea, pulmonary tuberculosis. No treatment. Disappeared.

CYSTS.

1. Gullion. Female, age 45. First seen in 1921. Gradual vision loss. Optic atrophy. In 1928, left-sided rhinorrhea. Laboratory test positive for cerebrospinal rhinorrhea. Left-sided paralysis. Narrow color fields. Last seen in 1935. Rhinorrhea still present. Condition attributed to intracranial cyst.

ETHMOIDAL INFECTIONS.

1. Safer. Female, age 45. Erosion cribriform plate. Frontal approach by Dr. N. Carter. Fascia inlay. No results.

2. Carmody. Female, age 40. Rhinorrhea. Ethmoidal infection. Seen with Dr. L. B. Lackard. Patient lost sight of for one and one-half years. Finally, death at Colorado General Hospital. Cause not given.

3. Daley. Female, age 32. Chronic ethmoiditis. Right-sided cerebrospinal rhinorrhea. Intranasal ethmoidectomy, neoprontosil. Head elevated. No intranasal medication. Recovery.

4. Thompson. Female, age 28. Rhinorrhea. Left-sided

chronic ethmoiditis. Polyps removed. Patient kept in bed two months. Rhinorrhea ceased after two weeks.

OSTEOMAS, FRONTAL AND ETHMOIDAL SINUSES – ARACHNOIDITIS.

1. Neuschatz. Case included in those of Dr. Davidoff.

2. Cody. Male, age 30. Seen 10 to 12 years ago. Rhinorrhea. X-rays showed osteoma which has perforated dura. Patient blew his nose, lost consciousness. X-ray showed air in cranial cavity. Patient developed brain abscess and meningitis. Death.

3. Bunts. Male in early thirties. Rhinorrhea associated with osteoma (intracranial) that extended into ethmoid. With pneumocephalus and X-ray findings, craniotomy was done. Osteoma removed and cribriform plate of ethmoid was covered with a piece of muscle. Several years later, patient developed meningitis and died.

4. Bunts. Female in early thirties. Rhinorrhea with communicating hydrocephalus. Probably due to an adhesive arachnoiditis. Another surgeon did a unilateral decompression. Patient got along well for a year or so following operation, then suddenly developed cerebrospinal rhinorrhea. X-ray of skull showed no pneumocephalus. Neurological examination was negative. Cerebrospinal rhinorrhea probably was the result of low grade increased intracranial pressure which resulted in dehiscence in cribriform plate of ethmoid. Decrease fluids and salt intake, plus lying horizontally in bed, did not help. Patient did not desire surgery. One and one-half years later, patient still has moderate rhinorrhea, although dangers of meningitis have been explained to her.

MENINGIOMAS.

1. Jordan. Female, age 47. Patient complained of headaches. Pain in right cheek and forehead and attacks of vertigo. Diagnosed as chronic hyperplastic ethmoiditis and maxillary sinusitis. Radical operation on right antrum, ethmoid and sphenoid on July 25, 1940. Several days later devel-

oped rhinorrhea. Subsequently complained of poor vision, although visual fields were normal. Eyes showed nystagmus to right and tongue protruded to right. Bilateral choked discs. Left palpebral fissure wider than right. Absent abdominal reflexes. Craniotomy done. Patient died within a few days. Meningioma had destroyed large portion floor right middle fossa and had invaded right sphenoid.

2. Raney reports a most interesting case: Male, age 48. History Jacksonian, convulsions on right side for nine years. History increased intracranial pressure for eight months when first seen by Dr. Raney. Had been blind for six months and comatose for two weeks. A large meningioma removed from left tempoparietal convexity of the skull. Patient made an excellent recovery, except for permanent blindness. One year later, patient returned complaining of cerebrospinal rhinorrhea. He was operated upon and a communication through the cribriform plate was found, the dural defect was closed, and there has been no return of the cerebrospinal rhinorrhea. Dr. Raney explained the cerebrospinal rhinorrhea as due to the long intracranial pressure which probably allowed erosion through the cribriform plate. As long as the intracranial pressure existed, the frontal lobe of the brain herniated through the defect and acted as a plug. After the meningioma was removed, the brain retracted and consequently the adhesions were broken and the cerebrospinal rhinorrhea resulted.

3. Van Wagenen. Two meningiomas. Age and details not given. Records not available. Physician in armed forces.

4. Allis. Reports an unusual case not strictly spontaneous cerebrospinal rhinorrhea, but having Paget's disease, which he thought caused softening of the floor of the anterior fossa. The side was much lower than the opposite side of the nose. The cerebrospinal rhinorrhea resulted from removal of hyperplastic tissue from the upper part of the nasal fossa. The opening was closed by a small piece of muscle.

PITUITARY TUMORS.

1. Guyton. This case reported in Dr. Shea's case.

2. Freedman. Female, age 58. At age of 15, headaches with cessation of menses. At age of 43, "catarrhal trouble," water came from both sides of nose, lasted two or three weeks. Treated with corpus luteum. At 50, short period of double vision. In 1941, X-rays showed erosion of left petrous area and posterior clinoids. Diagnosis, mass in region pituitary. Cerebrospinal rhinorrhea still continues. Headaches relieved when it occurs.

3. Pilcher. Male, age 40. Pituitary tumor with erosion sphenoid. Subtemporal decompression by another surgeon. Meningitis; death at home.

4. Harris. Two cases, females, ages 20 and 40. One case, left-sided cerebrospinal rhinorrhea, no cause found. Other, pituitary tumor. Both developed meningitis and died.

5. Silverberg. Female, age 44. Tumor pituitary. Treated with X-ray. Cerebrospinal rhinorrhea ceased. Well up to April, 1942.

6. Pace. Female, age 30. Benign neoplasm pituitary. X-ray showed increased size sella turcica and obesity. Developed meningitis, but recovered. Five years later, no other objective subjective sign other than continuous cerebrospinal rhinorrhea in varying amounts.

7. Coates. Female, age 45. Pituitary tumor. "Lost track of patient after two years."

8. Bunker. Female, age 63. Right frontal and temporal headaches. Right-sided cerebrospinal rhinorrhea. Confirmed by two laboratories. Fundi and visual fields normal. "X-ray showed erosion sella turcica especially posterior clinoids." Refused consultation with neurosurgeon. Condition same in June, 1944.

9. Howe. Male, age 52. Pituitary tumor operation by neurosurgeon. Death from shock 24 hours after operation.

10. Arrington. Female, age 50. Cerebrospinal rhinorrhea due to malignancy involving cerebrum, extended into nose,

involved septum. Too far advanced for surgery. Treatment with X-ray. Patient died.

LEPROSY.

1. Halahan. Female, age 26. Case seen in 1928. Anesthetic type. Not followed.

Major saw two cases, females, 25 to 30 years of age, in the Allegheny General Hospital, Pittsburgh, Pa. Records not available.

Golden reports two cases, females, ages 35 and 42. Cerebrospinal rhinorrhea still present. No treatment noted.

Tebbot. Female, age 25. Cerebrospinal rhinorrhea. Died after upper respiratory infection.

Robb. Female, age 38. In 1932, following a cold, patient developed cerebrospinal rhinorrhea. Lasted for 10 weeks from left nostril. In 1932 had a general check-up. Showed hypertension which she had had for six years. In 1935, patient suddenly gained 50 pounds in weight. Physical examination normal except male distribution of hair and other changes pointing to pituitary. Nasal examination revealed deviated septum, cloudy left antrum and left-sided rhinorrhea. Laboratory work negative, including blood counts, urinalysis, Wassermann and blood chemistry. X-ray of skull negative. When spinal tap was done, cerebrospinal rhinorrhea stopped, and commenced when stylet was placed again in needle. Patient died from meningitis, April, 1937.

Cannor reported one case that was seen years ago.

McKeowen. Male, age 56. Results good.

Torrell. Male, age 32. Cerebrospinal rhinorrhea diagnosed. Patient had been previously seen by New York otolaryngologists. "Wanted no treatment, simply desired to demonstrate he could fill test tube from drainage from left nostril."

Mancoll. Female, age 60. Cerebrospinal rhinorrhea. At first used some shrinking. Stopped all treatment and condition ceased spontaneously.

Weille. Female, age 40. Cerebrospinal rhinorrhea left side. End-results not given.

Voleman. Three cases. Ages 16, 24 and 30. Sex not given. All left alone. End-results good.

Tobey. One male and two females. One case hernation dura following ethmoid operation. Dehiscence proved at autopsy. Death from meningitis. One case cured by application 50 per cent silver nitrate.

Blaisdell. Female, age 42. Developed meningitis. Sulfa drugs used. Died four days after diagnosis of cerebrospinal rhinorrhea was made.

Rubenstein. Female in fifth decade of life. Methylene blue injected into spinal canal. Appeared in nose. Patient developed meningitis, but recovered. Finally lost sight of.

Wolfe. Female, age 60. Died of meningitis several months after diagnosis was made. Under care of another physician.

Arbuckle reports five cases. Ages from eight to 15. Three females and two males. All treated intranasally. End-results poor.

Myerson. Male in early forties. Patient observed for two years and then disappeared.

Fowler reports one case seen years ago in clinic. Cerebrospinal rhinorrhea gradually subsided.

Hambach. Male, age 78. General arteriosclerosis, hypertension and myocarditis. Death two weeks after first visit.

Kler. Male, age 25. Impression, "brain tumor." Patient, a transient, left city.

Zwerling. Male, age 55. Patient had multiple intraoperations elsewhere. Condition lasted several years. "Rhinorrhea from a drop to almost a flow on exertion."

Herbert. Female, age not given. Cerebrospinal rhinorrhea ceased after several months spontaneously. (Laboratory confirmation.)

Parnell. Female, age 45. Seen in consultation by Dr. Wells P. Eagleton. Refused surgery. Disappeared.

Beinfield. Young male. Spontaneous cure. No treatment.

Haney. Male, in late twenties. "Lost track of patient after six months."

Tribble. Female, age 50. No treatment. Death from "heart condition?"

Kettlecamp. Female, age 38. Seen in 1929. Complete recovery in 50 days.

Male, age ?, seen in 1937. Cerebrospinal rhinorrhea began six months ago. One year ago drained five or six days and stopped. Some history 20 years before. Patient lost sight of.

Bryant. Female, age 35. Seen in 1933 or 1934. Trichloracetic acid applied several times. Patient drifted into other hands.

Jones. Female in her thirties. Treated first intranasally in New Orleans, La. Cerebrospinal rhinorrhea ceased after two months' therapy. Recurred one year later and referred to neurosurgeon.

Chaffers. Two cases, male and female. No other data given.

Simpson. Female, age 70. Negative Wassermann. Skull X-ray. Stopped spontaneously two years after onset.

Berlin. Female, age 40. Patient died of coronary thrombosis.

Stonecypher. Female, age 36. Spontaneous cessation cerebrospinal rhinorrhea after six months.

Meyer. Male, age 18. Cerebrospinal rhinorrhea verified by laboratory examinations. Chemical cauterization middle meatus with trichloracetic acid. Recovery.

Lewis. Female, age 25 to 30. Sudden gush cerebrospinal fluid after headaches. Passed into other hands.

Murphy. Male, age 55. Cerebrospinal rhinorrhea attributed to a neoplasm. Died. Autopsy showed three typed malig-

nances: hypernephroma, neuoblastoma and carcinoma both lungs.

Bennett. Female, age 60. Seen prior to days of sulfa drugs. Upper respiratory infection and death from meningitis.

Smedley. Male, age 22. Cerebrospinal rhinorrhea cured by several cocainizations of sphenopalatine ganglion.

Lewy. Two females, past middle age. "One died of meningitis, other lost track of."

Morwitz. Female in her twenties. Laboratory substantiation. Ten per cent silver nitrate applied in olfactory area. Results satisfactory.

Wojniak. Female, age 45. "Lost sight of patient after two months."

Theobald. Two spontaneous cases: "In one case lipiodol was injected into spinal canal and shown by X-ray as streak in nose." Results in each case unknown.

O'Brien saw a female (age not given) 30 years ago. Condition has been present all her life. No treatment.

Semenov saw one case, but he is now in the armed forces, hence records are not available.

Lyons. Age and sex not given. One case cured by complete rest.

Blander. Two cases, age and sex not given. Clinic cases. Silver and other caustics used. No results. Patients disappeared before any surgery could be arranged.

Myers. Male, age eight. "Lost the case." End-results not known.

McNichols. Male, age 40; female, age 35; female, age four. Two treated by applications silver nitrate and recovered. One operated by a "brain surgeon." This case died.

Johnson. Two cases. One female, age four. Age of other not given. One case, no treatment. Followed for several

years. Other, fascia transplant by neurosurgeon but rhinorrhea persisted.

Haley. Female, age 50. Fascia implant over cribriform plate by neurosurgeon. Recovery.

Female, age 55. Right-sided cerebrospinal rhinorrhea for four and one-half years' duration. Developed severe meningitis, but recovered. Rhinorrhea ceased for several months, but now is worse than ever.

Jones. Male, age 22. Cerebrospinal rhinorrhea still persists.

Ruby. Male, age 21, and female, age 38. Cerebrospinal rhinorrhea still persists.

LeJeune. Two females, ages 35 and 65. Intracranial fascia transplants. Excellent results.

Lindsay. Female, age 40. Plastic flap dura by neurosurgeon. Cured of cerebrospinal rhinorrhea, but died from brain tumor. "The third ventricle had been blocked by the tumor, and the cerebrospinal rhinorrhea came from ventricular system."

Other case, "details not fresh in memory."

Breitstein. Two cases, males, 40 and 50. Laboratory confirmation of fluid. Both recovered spontaneously.

Keating. Male, age 50. Copper solution reduced. Spontaneous recovery in three weeks.

Miller. Male, age 45. Female, age 60. Condition continued. No treatment given.

Higbee. Female, age 40, who had seen many rhinologists for a right-sided discharge. Laboratory confirmed presence of cerebrospinal fluid. Operation by Dr. Howard. Naftziger, muscle transplant. Excellent results.

Parkinson. Female, age 45. Laboratory verification. Death one year later. Said by relatives to be "meningitis."

Wilkinson. Male, age 35, and female, age 45. One case recovered spontaneously, other died of meningitis.

Lemere. Two cases. Records lost. Both recovered. Cerebrospinal rhinorrhea ceased spontaneously.

Canfield. Three cases, adults, ages 20 to 60. Records not available. Dr. Canfield abroad in army. All were treated by intracranial fascia transplants with excellent results.

Soldiner. Female, age not given. Laboratory confirmation of cerebrospinal fluid. Successfully operated by neurosurgeon.

Hamby. Female, age 34. Onset at 33 years of age. Left frontal craniotomy. Dura sutured and muscles placed in sinus. Cerebrospinal rhinorrhea recurred in 18 months. Reoperated intradural closure of sinus with muscles and extradural waxing of bony defect. Patient still has occasional light drainage.

Bonvy. Three cases. One female, 42, and two males, ages 48 and 54. Treated with cautery trichloracetic acid and bed rest. Headaches when fluid stopped. Results not good.

Moffett. Female, age 48. Cerebrospinal rhinorrhea lasted two weeks, ceased and recurred in one year. Finally, after two years, ceased spontaneously.

Ingraham. Female, age 65. Patient seen only once. Had a severe hypertension with history of cerebrospinal rhinorrhea leak 12 years before, which ceased following tonsillectomy. Preparations for further study were being made when patient suddenly died. Autopsy findings not satisfactory so far as explanation of cerebrospinal rhinorrhea was concerned.

Weith. Two cases, females, ages 25 to 30. Records lost in moving. One case developed mastoiditis and was operated on. The cerebrospinal fluid gradually ceased during this period, although patient had drifted into hands of an osteopath.

The other case was taken over by family physician. Findings confirmed by laboratory test of fluid. Results not known.

Caron. Female, age 47. Seen in 1931. Complaints were of double vision and cerebrospinal rhinorrhea. Lost track of.

Female, age 58. Seen in 1942. Fifty per cent silver nitrate applied and after three months discharge stopped.

Anderson. Two females, ages not given. One had had condition recurring for a number of years and stopping. X-rays negative. The other came on suddenly. Both were treated by absolute bed rest, no coughing, sneezing, straining at stool, etc. Both recovered.

Means. Two cases; female, 40. Luetic. Other case, age not given. Both referred to neurosurgeon who did fascia implants over cribriform. Results not given.

Eagle. Male, age 40. Bed rest, dehydration sulfa drugs. Cured.

Ehrlich. Female, age 26. Bed rest for 96 hours and sulfa drug. Referring physician reported flow stopped after 48 hours.

Coleman. Six cases. Tumor on some cases and not known in others. (Records not available when questionnaire was answered.)

In all cases, cribriform fossae were blocked with muscle. Results excellent in two cases.

Ridpath. Male, middle life. Entirely recovered.

Stinson. Female, age 28. X-ray to pituitary, though no definite tumor noted. No rhinorrhea for over two years.

Orlando. In 1910 saw a male, 45 to 50 years of age. Clinic case who developed intralateral cerebrospinal rhinorrhea following a severe sneezing. Laboratory confirmation. Patient finally disappeared. Outcome unknown.

Dr. Selly, himself, has cerebrospinal rhinorrhea. Has practically stopped, although he has an occasional drip.

Jenkins. Female, age 53. Cause undetermined. Chief symptoms, headache. Recovery.

Buckman. Female, age 30. End-result not known. Referred back to family physician.

Teed. Female, age 48. At operation, smooth round hole, 2 mm. in diameter, in cribriform plate. No history of trauma or operations. Remainder area normal in appearance. Precipitating factor causing cerebrospinal rhinorrhea is not known. Patient had pneumonia twice prior to surgery. Osteoplastic flap, dura turned in and sutured. Recovery.

Wilkerson, W. W., Jr. Male, age 65. Cerebrospinal rhinorrhea continues.

Golden. Female, age not given. Cerebrospinal rhinorrhea followed an attack of influenza in 1918. Associated with the cerebrospinal rhinorrhea were disturbances of equilibrium. Last four or five years of life, discharge ceased. Died, coronary disease.

Cone. Female, age 36. Fascial transplant. Excellent results.

Hartsook. Male, age 45. Died, Female, age 30. Recovered.

Murphy. Female, age 38. Recovery after sodium iodide treatment.

Mood. Now in navy. Reports he had three cases of cerebrospinal rhinorrhea. Records not available.

Fergerson. Female, in middle twenties. Referred to neurosurgeon. End-results unknown.

Schintker. Female, age 40. Saw three years with spontaneous cerebrospinal rhinorrhea of six years' duration. Refused operation. Put to bed in semi-Fowler position. After two weeks, flow ceased.

White. Female, age 53. Cerebrospinal rhinorrhea finally stopped.

Cohen. Female, in her forties. Refused craniotomy. Lost sight of.

Sanders. Sex not given, age 47. Doubtful X-ray. Refused operation. Disappeared.

Shea. Three cases previously discussed. Dr. Shea says, "They rely on the use of a linen handkerchief test." The fluid is collected and allowed to dry in the handkerchief. If it is

smooth, the same as water, it is more than likely cerebrospinal rhinorrhea.

Equen. Male, age 36. Condition congenital affair. Cerebrospinal rhinorrhea persists.

Evans. Two cases, females, ages 43 and 30. In the first case, the cause is undetermined. Osteoplastic craniotomy and insertion of periosteal and fascial tissue over the cribriform plate. Results excellent. No recurrence after 16 months.

Second case suggested intracranial neoplasm, although not proven by ventriculography and encephalography. Same type surgery as in first case on two occasions, finally culture of pure staphylococcus. No results. Death from meningitis. Both sulfadiazine and penicillin therapy proved fruitless. Autopsy refused.

Falconer. Two cases. Sex not given, ages 30 and 55. Recognized at once. Both cases left for someone else.

Folk. Male, age 36. Recovered spontaneously.

Fuller. Saw one case 20 years ago. Cerebrospinal rhinorrea verified by laboratory. Case passed into other hands.

Shary saw one case years ago. Died from meningitis.

Strauss reports two cases, one male, 21, and female, age 40. Silver nitrate was applied to roof of nose without help. Discharge continues.

Harrell. Female, age 35. One per cent solution zinc sulfate applied in nose. Cured. Not seen after six months.

Walsh. Saw case spontaneous cerebrospinal rhinorrhea 10 years ago in Chicago. Records and other data not available.

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330 East Main Street.

AN EXPERIMENT SHOWING THE BERNOULLI ACTION IN EMPTYING THE NASAL SINUSES.

Z. WILLIAM COLSON, M.D.,

Lawrence, Mass.

After writing my paper on Bernoulli,[1] I have reviewed the chapters in Proetz's[2] "The Physiology of the Nose" which deal with pressures in the sinuses and find that my own experiments and those of Proetz are in complete agreement; in fact, if I had carefully digested all of Proetz's work before I began my experiments on Bernoulli, I would have expedited my investigations materially.

In order properly to introduce my experiment I shall review and slightly modify the convincing experiments of Proetz. The first three experiments are presented after the manner of Proetz, and each figure attempts to correlate its experiment with actual nasal conditions. In all cases, a section of glass tubing represents the nasal passage or airway, and the tap at the middle of the glass tube represents a nasal sinus ostium.

Since in all of these experiments inspiration produces suction at the tap, or sinus ostium, the arrows used in the illustrations show the direction of air flow on expiration only. Also, since we are interested in the Bernoulli effect in blowing the nose, we shall refer to forceful expiration in each case.

Fig. 1 shows a nasal passage with a large or normal choana and a constricted anterior naris. This may be a permanent constriction, such as a congenitally small nostril or an anterior septal deviation, or it may be temporary as in a local anterior engorgement of the turbinates. In either case, on forceful expiration, there will be a wide excursion of pressure registered in the nasal sinus. For convenience, we represent

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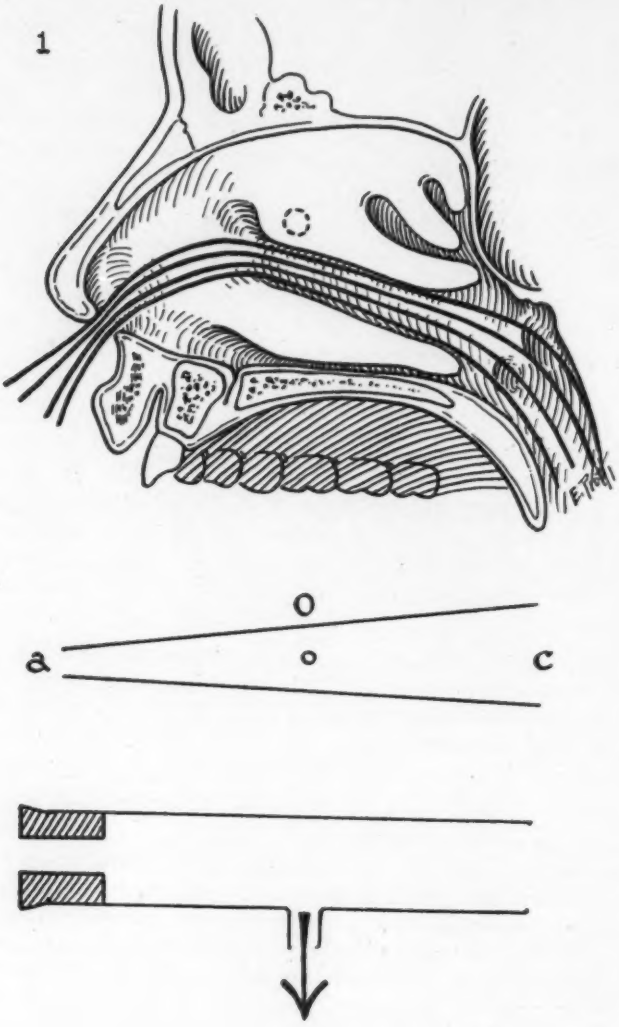

Fig. 1.

a sinus ostium placed at about the midpoint of the nasal breathing space opening into the maxillary antrum, but the same principle applies to any sinus.

The experiment which shows this is the glass tube, with middle tap, obstructed at its distal end by a perforated rubber stopper.

Fig. 2 represents obstruction at the choana, as with adenoid tissue, and is illustrated by simply reversing the ends of the stoppered tube. It is obvious that with a constricted choana and wide naris, the excursion of pressure in the sinus will be minimal.

Fig. 3 most nearly represents the normal nose with free choana and free anterior naris — although the dotted line is used by Proetz to remind us that in the normal nose the choana is slightly larger than the anterior naris. The experiment to illustrate it is the straight glass tube without stopper. On forceful expiration there is a moderate increase of pressure in the antrum.

Fig. 4 shows what we can expect when the constriction is not anterior or posterior, but is in the region of the ostium itself. This is represented by a drawn glass tube forming a Venturi constriction with a tap at the constriction. When we blow through this tube the water manometer shows definite suction. This is the condition which we find in the nose when the turbinates become congested and swell into close apposition in the region of the ostium.

This glass Venturi tube was made for me by Dr. Morton Grant, of the Howe Research Laboratory, Harvard University. The following is his brief description of how he did it: "Glass models were constructed by heating and drawing-down glass tubing of 10 to 20 mm. diameter to produce a symmetrical constriction. At the narrowest point a small hole was blown in the side-wall and a glass tube of 1 to 4 mm. diameter fused into it."

If, however, you wish a simpler apparatus for showing the

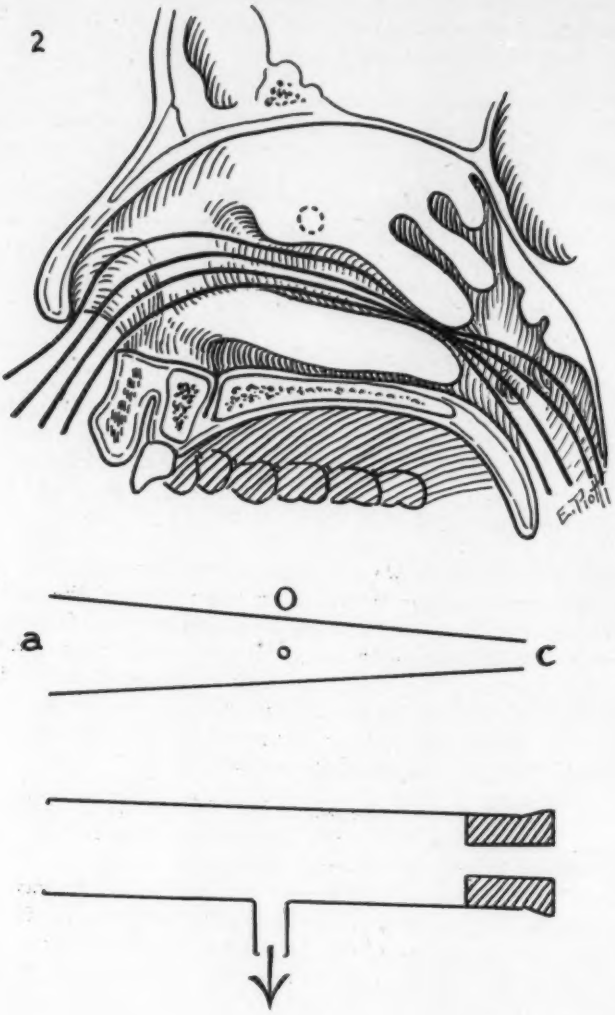

Fig. 2.

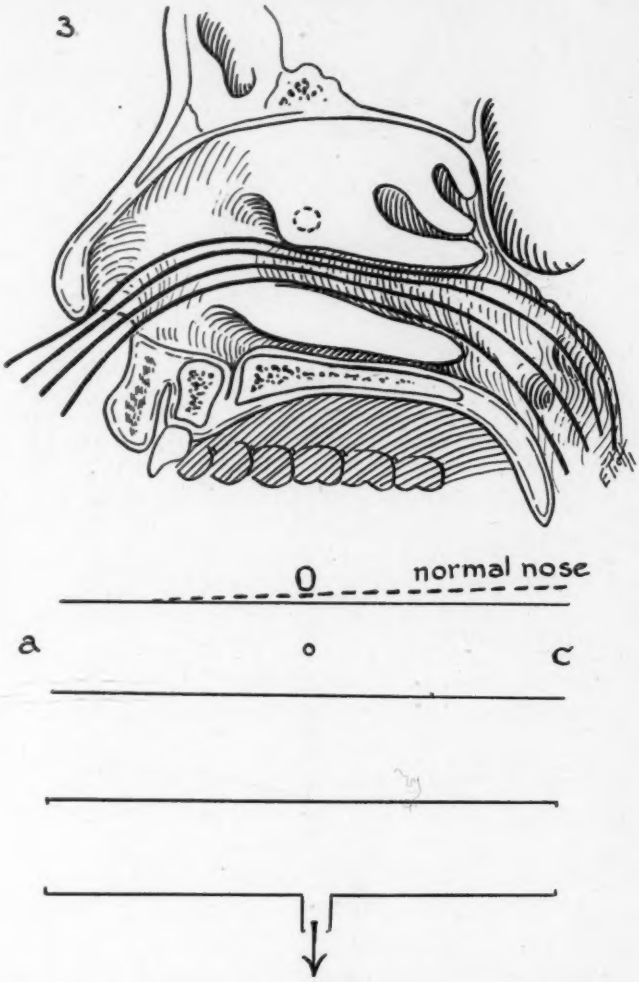

Fig. 3.

Bernoulli effect in the nose, beg or borrow an old common duct T-tube from your operating room supervisor. Insert an

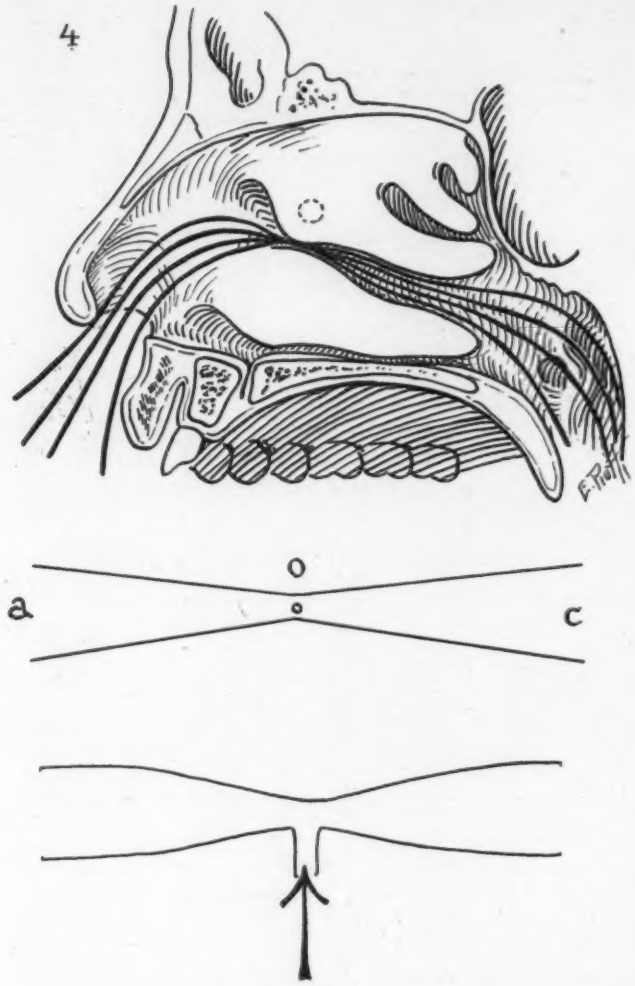

Fig. 4.

ordinary medicine dropper into the cross of the T, so that the point of the dropper is near the opening in the leg of the T.

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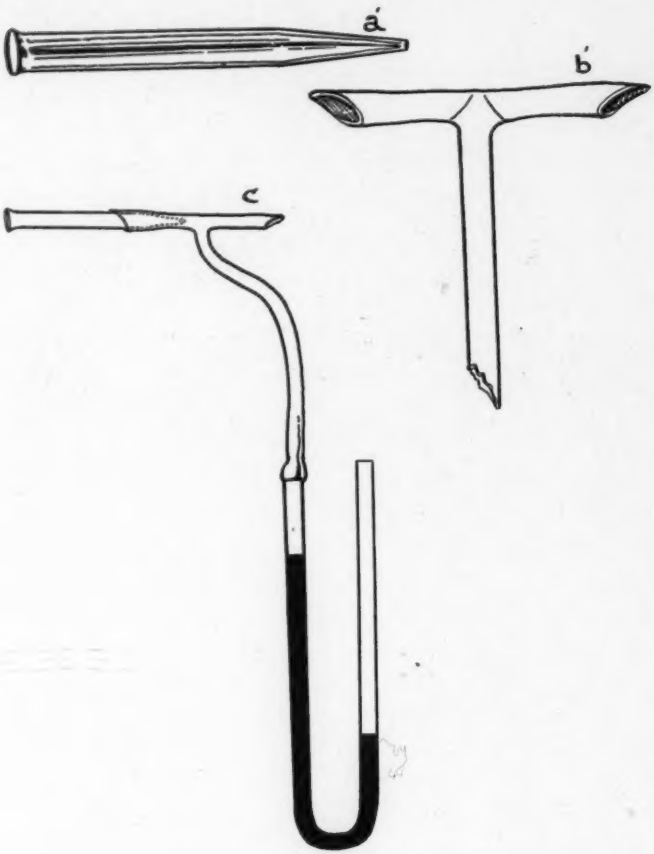

Fig. 5.

Connect the leg of the T to a water manometer. Now forcefully expire and inspire through the medicine dropper. You will note that under both conditions the water manometer registers suction (see Fig. 5).

The primary appeal of the latter experiment is its simplic-

ity. It is not as accurate a reproduction of the streamlined constriction which neighboring soft tissues produce when they expand toward each other. Also, since it is in effect only the first half of a Venturi tube, it lacks the latter's efficiency. Its materials, however, are readily available in any community, and it is simple to put together.

It is commended to anyone who wishes to demonstrate for himself the Bernoulli action in a congested nose.

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301 Essex Street.

IN MEMORIAM

ISIDORE FRIESNER, M.D.,

1874-1945.

Dr. Isidore Friesner, aged 71, died on Sept. 8, 1945, at his home in Katonah, N. Y., of coronary artery disease.

He received his B.A. degree at the College of the City of New York in 1894, and his M.D. degree at Gross Medical College in Denver, Colo., in 1901.

Upon his return to New York, he became affiliated with the Manhattan Eye and Ear Hospital and the Post-Graduate Medical School where he taught otologic technique. At the Manhattan Eye and Ear Hospital he was associate surgeon on the Wendell Phillips Service until 1919, when he resigned to accept the position of otologist to Mount Sinai Hospital, New York, succeeding Dr. Frederick Whiting. While at the Manhattan Eye and Ear Hospital he wrote two books in collaboration with Dr. Alfred Braun, "Cerebellar Abscess" and "The Labyrinth." His contributions to otologic literature were numerous and varied, indicating a keen interest in the allied basic sciences.

He was a member of the editorial board of the *Archives of Otolaryngology* for 20 years, having been one of the original board at the inception of that journal. In recognition of his outstanding ability, he was elected president of the American Otological Society in 1939, and secretary-treasurer of the Society from 1940 to date. He was a member of the American Laryngological, Rhinological and Otological Society, Inc., the American College of Surgeons, the American Medical Association; vice-president of the New York Otological Society, New York Academy of Medicine, New York State and County

ISIDORE FRIESNER, M.D.

Medical Societies, and was a former vice-president of the New York League for the Hard of Hearing.

He was consultant otologist to Mount Sinai Hospital, Beth David Hospital, Bronx Hospital, Methodist Hospital, St. Joseph's Hospital, Far Rockaway, and Flushing Hospital, Flushing, N. Y. He was chairman of the Medical Board of Mount Sinai Hospital for 12 years, in which position his foresight and judgment were of inestimable value to the welfare of the hospital.

His outstanding personality and charm, his sustained interest in clinical otology and histopathology, neuro-otology, bacteriology and internal medicine served as an inspiration to the numerous young men whom he trained and with whom he came in contact.

The community in which he practiced has lost an eminent otologist, Mount Sinai Hospital one of its most respected sons, and we who have been intimately associated with him have lost a staunch friend and an inspirational leader. H. R.

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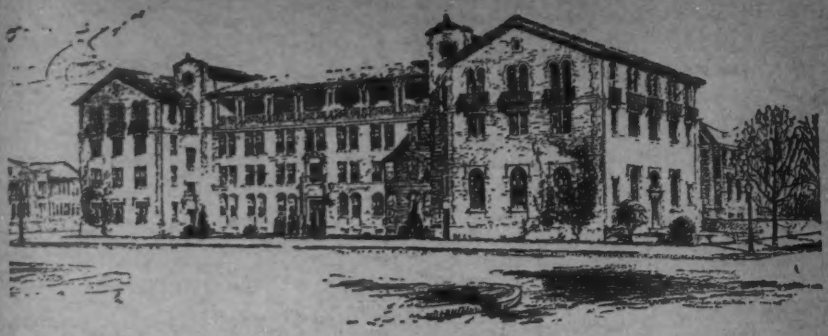

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